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DISEASES OF THE NERVOUS SYSTEM

A PRACTICAL JOURNAL ON PSYCHIATRY AND NEUROLOGY

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DISEASES of the NERVOUS SYSTEM

(PUBLISHED MONTHLY)

A PRACTICAL JOURNAL ON PSYCHIATRY AND NEUROLOGY

Prognosis in Some Mental Disorders

LEWIS DANZIGER, M.D.*
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When a new treatment is proposed, its results must be judged by comparison with old treatments. This raises questions which can be answered only quantitatively. Among other things, we desire to know of the new treatment: do more patients recover under it?, do they recover more quickly or safely?, is the treatment truly economical or can it only be called cheap or expensive?

The need for such information has been well illustrated by the continuing uncertainty as to the value of shock therapies. After more than nine years experience in the United States, there is still wavering and in the discussion of the matter, bitterness and passion may still be observed. Although the survey of Kolb and Vogel[1] showed the use of shock therapy was wide spread and increasing, doubt as to its real value continue to be expressed in the literature and in private discussions. A large part of the uncertainty appears to exist because no standard tables of prognosis are readily available for the principal psychoses.

We should have tables for a year prior to 1936 (when insulin therapy was introduced in the United States) showing:

1. The per cent of patients who recover or improve in the hospitals.

2. The duration of hospitalization before the change is observed.

These data will answer questions such as the following: of 100 patients with dementia praecox admitted to the hospital, how many will go home recovered within three months?, within six months?, within five years? How many will go home improved in any given interval? The answers to such questions may be derived from the tables presented in this paper.

The U. S. Bureau of the Census[2] has prepared and published an extensive study of the movement of state hospital population for the year 1933. The raw data of this survey furnish the basis for the present investigation. The report for 1933 was selected because:

1. In that year, symptomatic and other treatment of pre-insulin shock times had reached a high state of development.

2. The survey covers the state hospitals of the United States, which in 1933 contained about 87 per cent of all the beds available in this country for the treatment of mental disorders and which admitted in 1933 about 69 per cent of all mental patients in this country. The results obtained in state hospitals may have been better or worse than those obtained in other institutions, but they were the results available to the majority of the patients.

3. The reports for 1933 were filed before most American state hospital psychiatrists were aware of the work of Sakel and Meduna and hence before their opinions as to prognosis could have been affected (if they were) by bias for or against shock therapy.

The Census reports show:

1. The number of first admissions and readmissions by sex and psychosis.

2. The number of patients of each sex and psychosis who left the hospital as recovered or improved.

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3. The duration of hospitalization of each patient who went home.

From these data it is possible to calculate for each interval of the period of hospitalization the percentage of patients discharged as recovered or improved. The calculation may be illustrated by the following example for dementia praecox. First admissions plus readmissions totalled 18,877. In the first month of treatment, 12 of these patients were discharged as recovered. The number recovered per 100 admitted is for this interval (the first month) then,

$$\frac{12 \times 100}{18{,}887} = 0.06$$

In the second month of treatment, an additional 61 patients were discharged as recovered. The number recovered per 100 admitted for this second interval then is,

$$\frac{61 \times 100}{18{,}887} = 0.33$$

And so on for each other interval and for each type of disorder, whether the patient was discharged as recovered or improved. The divisions were done by slide rule and the quotients are reported to the second decimal place. This method leads to columns 1 and 3 in Tables 2 to 5.

The total of column 1 shows, for the whole period of hospitalization, more than 20 years,

TABLE 1
PATIENTS ADMITTED TO STATE HOSPITALS IN 1933

	MALE			FEMALE		
Diagnosis	*Admitted*	*Readmitted*	*Total*	*Admitted*	*Readmitted*	*Total*
Dementia praecox	7518	2621	10139	6384	2354	8738
Manic-depressive psychosis	4020	2029	6049	4917	2728	7645
Psychoneurosis and neurosis	690	128	818	901	215	1116
Involutional psychosis	446	96	542	916	191	1107

TABLE 2
PROGNOSIS IN DEMENTIA PRAECOX IN 1933 IN STATE HOSPITALS

Duration of hospitalization in months	*Discharged recovered in interval per 100 admitted*	*Probability of eventual discharge recovered which exists at end of interval*	*Discharged improved in interval per 100 admitted*	*Probability of eventual discharge improved which exists at end of interval*
on admission	...	0.0545	...	0.3240
0 to 0.9	0.06	0.0539	0.62	0.3178
1 to 1.9	0.33	0.0506	2.08	0.2970
2 to 2.9	0.43	0.0463	0.80	0.2890
3 to 3.9	0.53	0.0410	4.20	0.2470
4 to 4.9	0.49	0.0361	2.56	0.2214
5 to 5.9	0.42	0.0319	2.06	0.2008
6 to 6.9	0.33	0.0286	1.90	0.1818
7 to 11.9	1.03	0.0183	5.80	0.1238
12 to 23.9	0.88	0.0095	5.38	0.0700
24 to 35.9	0.34	0.0061	2.40	0.0460
36 to 47.9	0.16	0.0045	1.16	0.0344
48 to 59.9	0.08	0.0037	0.59	0.0285
60 to 119.9	0.14	0.0023	0.60	0.0125
120 to 239.9	0.07	0.0015	0.65	0.0060
240 and over	0.03	0.0012	0.20	0.0040
unknown	0.12	.....	0.40	
TOTALS	5.45		32.40	

the number recovered per 100 admitted. In the case of dementia praecox, this was 5.45. For each 100 patients admitted because of this disorder, therefore, 5.45 will eventually be discharged as recovered. "Eventually" it should be emphasized, may mean after 20 years or more of hospitalization.

The derivation of the second column may be seen from the following example. The first column shows that of the patients admitted because of dementia praecox, 5.45 per cent will recover eventually in the hospital. (An additional undetermined number may leave the hospital and recover at home). Any individual patient, selected at random from the dementia praecox group, then has on the day of admission a probability of eventual recovery in the hospital given by

$$\frac{5.45}{100} = 0.0545$$

(As is usual in calculations of probability, unity represents certainty and zero represents impossibility). It is thus seen that each patient with dementia praecox has, on the day of admission, a probability of eventual recovery in the hospital which is small but not infinitesmal.

In the first month of hospitalization, 0.06 of the 5.45 patients who will eventually recover, do recover and leave the hospital. Any individual member of the original group who remains then has left to him a probability of eventual recovery in the hospital given by

$$\frac{5.45 - 0.06}{100} = 0.0539$$

In the second month of hospitalization, an additional 0.33 of the 5.45 patients recover and leave the hospital. Any individual member of the original group who remains then has left to him a probability of eventual recovery in the hospital given by,

$$\frac{5.45 - (0.06 + 0.33)}{100} = 0.0506$$

TABLE 3

PROGNOSIS IN MANIC DEPRESSIVE PSYCHOSIS IN 1933 IN STATE HOSPITALS

Duration of hospitalization in months	*Discharged recovered in interval per 100 admitted*	*Probability of eventual discharge recovered which exists at end of interval*	*Discharged improved in interval per 100 admitted*	*Probability of eventual discharge improved which exists at end of interval*
on admission	...	0.3729	...	0.2480
0 to 0.9	1.04	0.3625	1.01	0.2379
1 to 1.9	4.16	0.3209	2.58	0.2121
2 to 2.9	4.26	0.2783	2.47	0.1874
3 to 3.9	4.51	0.2332	2.58	0.1616
4 to 4.9	3.56	0.1976	2.11	0.1405
5 to 5.9	2.73	0.1703	1.53	0.1252
6 to 6.9	2.16	0.1483	1.37	0.1115
7 to 11.9	5.49	0.0938	4.10	0.0705
12 to 23.9	4.80	0.0458	3.60	0.0345
24 to 35.9	1.55	0.0303	1.07	0.0238
36 to 47.9	0.80	0.0223	0.58	0.0180
48 to 59.9	0.37	0.0186	0.40	0.0140
60 to 119.9	0.67	0.0119	0.50	0.0090
120 to 239.9	0.20	0.0099	0.25	0.0065
240 and over	0.08	0.0091	0.07	0.0058
unknown	0.91	.....	0.58	.....
TOTALS	37.29		24.80	

And so on for the rest of column 2. Column 4 is derived in a precisely similar manner.

It is clear from the tables that the probability of recovery in the hospital declines rapidly with the passage of time until it approaches, in the case of dementia praecox, a value of less than 0.01, after which the rate of decline is less. The odds against recovery in the hospital are seen to be, on admission, 10,000 to 545; after about 20 years, they have risen to 10,000 to 15.

The tables have the immediate utility of furnishing a set of data about a control group, for assessing the value of new treatments. Columns 1 and 3 show the per cent of patients who will recover or improve in any specified interval. Columns 2 and 4 show the probability of eventual recovery or improvement remaining at the end of each interval.

The criteria for treatment more effective than the 1933 variety are:

1. A higher incidence of recovery or improvement, or
2. A shorter period of treatment necessary to produce results, or
3. Both 1 and 2.

If a treatment is found to be effective, its cost in labor and material may readily be compared with the cost of maintaining patients in the hospital for the period necessary for equal improvement under other types of therapy. In another communication, these criteria will be applied to shock therapy in dementia praecox.

The tables also furnish hospital staffs with a standard against which to check their own work. If modern therapeutic methods are used, there should be no reason for a hospital to fall below the 1933 standard.

The sex of the patient makes a small difference in the prognosis, not shown in the present report. This difference is greatest in the psychoneurotic and involutional groups,

TABLE 4

PROGNOSIS IN INVOLUTIONAL PSYCHOSIS IN 1933 IN STATE HOSPITALS

Duration of hospitalization in months	*Discharged recovered in interval per 100 admitted*	*Probability of eventual discharge recovered which exists at end of interval*	*Discharged improved in interval per 100 admitted*	*Probability of eventual discharge improved which exists at end of interval*
on admission	...	0.2090	...	0.2330
0 to 0.9	0.03	0.2087	0.545	0.2275
1 to 1.9	1.79	0.1908	1.80	0.2094
2 to 2.9	2.06	0.1702	2.67	0.1827
3 to 3.9	1.94	0.1508	1.77	0.1650
4 to 4.9	2.06	0.1302	1.25	0.1525
5 to 5.9	1.27	0.1175	1.77	0.1348
6 to 6.9	1.09	0.1066	1.63	0.1185
7 to 11.9	3.81	0.0685	3.50	0.0835
12 to 23.9	3.55	0.0330	3.90	0.0445
24 to 35.9	1.00	0.0230	1.15	0.0260
36 to 47.9	0.50	0.0180	0.70	0.0185
48 to 59.9	0.55	0.0125	0.75	0.0120
60 to 119.9	1.05	0.0020	0.65	0.0095
120 to 239.9	0.20	.....	0.15	0.0080
240 and over	0.00	.....	0.05	.....
unknown	0.00	.....	0.75	.....
TOTALS	20.90		23.30	

but it is probably not of importance for most clinical purposes since any new treatment for which success is claimed should give results vastly superior to those obtainable in 1933. The effect of sex can readily be calculated from the original census tables.

Probably more important than sex are the age of the patient and the duration of the illness before hospitalization. These quantities unfortunately are not available at present.

It should be emphasized that the material of this report concerns only state hospital patients. The results for private and teaching institutions might turn out to be considerably better.

Summary

From raw data of the U. S. Bureau of the Census, there have been prepared tables to show the prognosis for state hospital patients classified in four nosological groups. The prognosis was calculated for the year 1933 for comparison with the results of shock therapy and other types of treatment introduced since 1933. The tables show the chance of discharge recovered or improved in any portion of the first twenty years of hospitalization.

The chance of recovery becomes very small after two or three years of hospitalization, but some chance of improvement remains even after ten years of hospitalization.

References

1 Kolb, L., and Vogel, V. H.: "The Use of Shock Therapy in 305 Mental Hospitals," *Am. J. Psychiat.*, 99: 90, 1942.

2 U. S. Bureau of the Census: "Patients in Hospitals for Mental Disease, 1933," *U. S. Govt. Print. Office*, Washington 25, D. C., 1935.

TABLE 5

PROGNOSIS IN PSYCHONEUROSES AND NEUROSES IN 1933 IN STATE HOSPITALS

Duration of hospitalization in months	*Discharged recovered in interval per 100 admitted*	*Probability of eventual discharge recovered which exists at end of interval*	*Discharged improved in interval per 100 admitted*	*Probability of eventual discharge improved which exists at end of interval*
on admission	...	0.2840	...	0.4090
0 to 0.9	2.01	0.2639	5.07	0.3583
1 to 1.9	4.44	0.2195	7.83	0.2800
2 to 2.9	3.55	0.1840	5.78	0.2222
3 to 3.9	3.55	0.1485	4.17	0.1805
4 to 4.9	1.85	0.1300	2.65	0.1540
5 to 5.9	1.22	0.1178	1.92	0.1348
6 to 6.9	1.43	0.1035	1.68	0.1180
7 to 11.9	3.25	0.0710	4.50	0.0730
12 to 23.9	4.50	0.0260	4.10	0.0320
24 to 35.9	1.20	0.0140	1.10	0.0210
36 to 47.9	0.40	0.0100	0.24	0.0186
48 to 59.9	0.20	0.0080	0.36	0.0150
60 to 119.9	0.35	0.0045	0.50	0.0100
120 to 239.9	0.05	0.0040	0.10	0.0090
240 and over	0.00	.....	0.00	.....
unknown	0.40	.....	0.90	.....
TOTALS	28.40		40.90	

Equilibrium Neurosis

(Studies of Occupational Neuroses, No. 3[1,2])

ERNEST HARMS
New York, New York

For several years I have been collecting material on a rare form of neurosis which has been on the increase and on which I have been unable to find any comments in the literature. I should like to call this neurosis the "Equilibrium Neurosis," the reason for which designation will be apparent from the following description: The somatic picture correlated to this mental disease is of such a character that it must be classified as an occupational disease.[3] Theoretically this form of occupational disease results from the monotony of an occupation which forces the afflicted person to try to establish some kind of equilibrium. This may lead to a mental conflict and ultimately to a more or less severe neurosis.

I want to illustrate this conception by describing some cases which demonstrate my point of view:

The first case is that of an undertaker who, after working hours, seeks gay company and manifests an especially loud and boisterous behavior. His laughter is hysterical and his jokes seem forced. When questioned he explains: "I get fed up with playing the role of the mourner day in and day out. In order to endure it, I have to seek gay company in my leisure time." But he cannot continue this dual life for a long time. Some months after my first meeting with the man, a social one, he came to me in a state of complete mental breakdown. He could no longer continue the mourning attitude required of his profession and he became aware of an inner urge to be gay, even within his working hours. He noticed that he wanted to burst into laughter when he should have been extremely somber. He had developed an anxiety neurosis, being afraid that he might be unable to control his impulse to laugh. These signs of compulsive laughter made him realize that he was sick and needed psychiatric assistance.

This case reveals a clue to the basic character of an equilibrium neurosis. Because of his disposition, the man is unable to put himself permanently into the forced emotional status of perpetual mourning. His natural psychic inclination reacts against such an unnatural part and through compensating dynamics he produces this reaction which we may call a "Psychic equilibrium": the attempt at an outlet in the emotionally opposite direction of an equally exaggerated gay activity. But such gaiety is not in accordance with his occupational sphere and the more he tries to restrict his reactions to mournful gestures, the more does he increase the inner conflicts, which finally tend to endanger his entire professional activity. This leads to a severe neurotic condition.

There are a great many professions which create similar conflicts with the danger of causing mental pathologies. Some time ago I brought this problem to the attention of the psycho-therapists by an example of the medical profession itself.[2] I stressed the fact that there may be rather tragic psychological situations connected with medical practice that may produce pathological symptoms even during the first years of medical school. Many young men want to become doctors for idealistic reasons, to relieve suffering and heal the sick. But as soon as they start their courses in anatomy and become acquainted with the realistic and often gruesome facts of medical practice, their idealism breaks down and as a reaction they often develop an attitude of cynicism which may be expressed in crude jokes. When I was a student we became aware of this behavior of our medical colleagues and called their jokes "medicynical," knowing that the attitude had its roots in the psychological background of their choice of profession. When their studies are completed and they engage actively in their profession, these men usually develop a realistic concept of life.

However, this does not always develop in a smooth healthy manner. Jarl Hemmer, the Finnish novelist, in his book,

"The Fool of Faith" related the story of a medical man who, being sensitive as a boy, suffered severely in the early stages of his career. He tried to balance his emotional reaction to medicine by listening to music. But a fast developing neurosis interfered with his enjoyment of music, especially during concerts. He could not concentrate on the performance, for while watching the audience he would be obsessed in contemplation on the digestion of the people or the variety of facial expressions. If he fell in love with a girl, thoughts about the medical aspects of the perspiration underneath her pretty curls killed all erotic feelings. This severe neurosis made him a fugitive from human society and he wound up as a physician in a penal institution, living under horrible conditions. In his leisure he sustained his emotions by playing the violin, incessantly repeating Cesar Frank's "Andante Religioso". This made it possible for him to endure his environment. He developed an equilibrium which served its purpose.

However, even such a crude equilibrium cannot always be achieved. I have collected cases of medical men whose lack of equilibrium led to mental tragedies. Of other cases which I was able to personally observe and follow, was one of suicide and another which ended in morphinism.

Pronounced equilibrium neuroses may often be observed in professions concerned with enforcement of morals and ethical laws, e.g. that of the lawyer and the theologian. Members of these professions have frequently been chosen as heroes of books in European literature. There is the minister whose duty it is to teach ethics and abhorrence of sin, and there is the lawyer or judge or prosecutor who is supposed to bring the criminal to trial, but who finds himself in an unlawful situation. Scandinavian, German and French authors have used these tragedies of modern civilization as material for impressive poetic works.

To follow through my theme I describe a case from my own experience: It is that of an attorney, well known for his interest in his profession, and his progressive attitude. An ardent sportsman and hunter, he had a passion for auto races. Occasionally, at parties, he would imbibe heavily endeavoring to excuse himself jokingly saying: "It is not bad to commit a little sin once in a while". Before I met him personally, a mutual friend told me that he gave as a reason for his alcoholic excesses the need to counterbalance his regular occupation of enforcing the law, by committing an "innocent sin" once in a while. This is, no doubt, one of the more tragic forms of equilibrium neurosis. In the development of the neurosis, our attorney tried to reestablish his equilibrium by indulging a desire to break traffic laws. But the neurosis progressed rapidly toward a catastrophe. In his neurotic state he became uncertain of his ability to drive a car. After a minor accident caused by this feeling of insecurity, he developed a serious anxiety neurosis, ending in a kind of claustrophobia. He finally became unable to drive a car and showed pathological fear of speed, even when riding on a train or bus.

The following tragic example involved a member of the clerical profession:

It is based on a short note written by a young clergyman a few hours before he committed suicide. He had been a student of law when he fell in love with a married woman. He developed a state of ethical anxiety which caused him to change his intended profession and to become a clergyman. Shortly after he had been ordained the woman reappeared in his life. Unable to resist her, he again succumbed to her charms. Every day as he stood at the alter, a vision expressing his sinful desire for that woman appeared before his eyes and made it impossible for him to function in his profession. He developed a severe melancholia which lead to a complete nervous breakdown and ultimately to suicide.

All the instances of equilibrium neurosis, which I have described come under the main heading of social psychiatry.[4] I shall, therefore, in introduction discuss the social-psychiatric aspect of this form of mental disturbance. Put briefly, social psychiatry studies the environmental causes of psychopathologies. To what extent do social and environmental factors represent direct or indirect causes of mental disturbance and to what extent can they become factors in healing? These environmental factors are of utmost importance to psychopathology and

therapy of neuroses, as well as psychoses. Occupations are social activities and a person's occupational sphere constitutes his social environment.

The following illustrations will make clear this point of view: If a worker in a munitions factory where an explosion occurs through the mistake of a fellow worker is physically and mentally injured, the accident and its consequences are the result of environmental circumstances. The direct possibility of our individual preventing the accident can be ruled out. Even his indirect responsibility can hardly be considerable. One may say that people should exercise more caution by choosing less hazardous trades or professions. Technically every occupation has hazards. This problem has still other aspects, as was so ably demonstrated by Charles Chaplin as the factory worker in "Modern Times". In that film the little laborer cannot adapt himself to the modern super-technical conditions of his work and becomes more and more confused, irritated and disturbed until he breaks down.

From the viewpoint of individual psychiatry we might reason: an individual who cannot stand modern work methods is in himself a weak, neurotic and underprivileged person, and a breakdown should not be attributed to environmental factors, but to the pathological tendencies of the individual. We could even go further: If a person comes in contact with a social situation, which he is constitutionally unable to endure, he must try to find some way to get over and out of this situation which endangers his mental health. Social organization, however, in our times is no less complicated than is a battle field in modern warfare. One must, therefore, regard such individual psychiatric motivations just as one-sided as the famous discussion on "our neurotic times," a slogan which blames all neuroticism on the social environment which we have created and in which we have been living more or less happily for a long time.

I do not overestimate environmental and social causes as the basis for mental abnormalities. But looking over the field of psychiatric observation we note a serious underestimation of social causes. Many of our neuro-physiologically oriented psycho-therapists maintain that environmental circumstances play only a "precipitating role in psychoses". This reasoning is just as fallacious as an opinion that every child is born with a healthy nervous system, but is made sick by its environment. I want to advocate a more correct and more sincere observation and judgment of the social conditioning of mental disease.

This wide spread misunderstanding of the social background of mental disease is not caused by the irresponsibility of certain psychiatrists, but is inherent in most of our present day psychiatric thinking, which has not yet learned to include social processes and social aspects in its reasoning. Most psychiatrists will agree with the generally accepted and fundamental principle that we can only heal a disease if we know its cause. This principle was first adopted in bacteriology, but it is not less true in psychiatry. So far the majority of psycho-therapists are not concerned with the causes which brought about this or that mental disturbance,—causes, which might lie far back in the patient's early life and environment. Many psychiatrists confine themselves to the somatic findings which can be observed. These findings are regarded as the denominator of the somatic pathological picture for which prescriptive measures are taken. This presents a medical picture in which the individual cause, creating the disturbance, has no place. In the so-called "Individual Psychiatry" the individual as a whole, the entirety of his existence as related to his disease are not considered but rather the individual is regarded as "a case of," on a textbook classification of mental illnesses. If they would view the patient in his totality, they would have to include a more careful consideration of the causes of the specific difficulty which would lead beyond the office or the institutional aspects.

C. J. Jung introduced the observation of the final and causal aspects of a mental disturbance into psychiatric observations, and has demonstrated their close inner relationship. Only a sincere and exhaustive study of the causes may lead to improved therapy in mental disease. Alfred Adler also emphasized the need to study the causative factors of a pathologic personality. However, we are still

far from having a clear understanding of the social components of mental disease, even those of a merely accidental character. For many years I have attempted to discover certain categories of social influences which might be considered primary conditions, as in equilibrium neuroses.

From the psycho-pathological angle, it can be said that an equilibrium neurosis may occur in connection with any occupation. Of course, every mental illness stemming from an occupation is not necessarily an equilibrium neurosis. A simple breakdown under mentally unfavorable conditions as presented in the Chaplin film is not an equilibrium neurosis. The specific factor in this disease is some kind of an attempt to escape into an opposite position, as from mourning into amusement, from law enforcement into crime, from ethical pursuit into immoral action. One could add many more instances, as from the exactness and accuracy of an accountant to deliberate mistakes, falsifications or frauds.

The Swedish psychiatrist, Paul Bjerre,[5] in his book on the millionaire swindler, Ivar Kreuger, portrays the young student's unusual abilities as a pupil, who, nevertheless, liked to fail in class because school was too boring for him and he enjoyed the excitement of cheating. There are frequently, people in high public authority who enjoy being ordered around by their babies. History is full of examples of crimes caused by tendencies toward an equilibrium neurosis. An artist or craftsman may enjoy to produce trash, only to live in fear that these unartistic products will become a threat to his reputation.

Looking at the first two examples, that of the lawyer and the minister, we may be led to assume that an equilibrium neurosis is based primarily on a moral conflict. However, this is not always the case. There may be a multiplicity of reasons involving the occupational activity in a direct or in an indirect way. Directly in the disqualification or even the loss of the profession itself, or indirectly through the fear of the loss of position or social prestige. Such an equilibrium neurosis is always based upon a conflict of the individual in his relationship to the profession.

A second point to be considered is the motive leading to the conflict. It must necessarily be of a negative nature. Dissatisfaction with the profession must exist. In regard to the social-pathological situation we have to consider the motive which brought the individual into his profession but later involved him in a conflict. The young clergyman who changed his profession because of ethical scruples is illustrative of this. Frequently financial or social pressure is the motive in selecting a profession not in accord with the individual's inclination. This factor is of major importance in our later social-therapeutic considerations. While dissatisfaction and frustration are complicating factors, minor dissatisfaction alone will not lead to an equilibrium neurosis. In present day occupations there are many individuals who do not actually like the work they are engaged in, but only a small percentage attempt an escape in the form of an equilibrium. This leads to the question: What is the specific character of an occupational situation resulting in an equilibrium neurosis?

If we examine the various forms of such a neurosis, we can make the following observation: In none of the examples given did the afflicted person want to escape from his profession. He may be "sick of it" or "tired of it," he may even hate it and prefer suicide to a healthier solution of an unhappy life-situation, but he refuses to relinquish his profession, clinging to it by sheer will. Equilibrium thus becomes an action within the life sphere of a professional activity. It is characteristic of an equilibrium situation that the individual believes that he must remain within the unhappy professional sphere. From this fact arises the second specific element of his neurosis. Because the individual feels that no other escape is open to him, he develops a resistance against his activity which leads to a complete confusion between right and wrong in the profession. If the man would relinquish his occupation, he could easily neutralize his hostility and he would not be forced to extreme attitudes and inner animosity towards his own activity. In this respect the neurosis resulting from the equilibrium situation is the most specific professional neurosis and also the most difficult one, as far as therapy is concerned.

In social-psychiatric consideration of the equilibrium neurosis, we must emphasise the

fact that the individual concerned always takes his profession seriously. Over and above the conventional attitude towards it, as his chosen occupation, he feels that this is the only possible field for his professional activity. Whether or not the reason for this lies in social pressure or in the individual proper, is not significant.

Before proceeding to individual psychiatric considerations, I wish to refer to a general psychological viewpoint which will help considerably in understanding the dynamics of the neurotic state. We have seen that it is specific for the genesis of the equilibrium situation that the person involved develops a negativistic attitude toward his professional sphere, which, under normal conditions, would be a positive one. This is the psychodynamic factor appearing in every case of this neurosis. Our emotional life is characterised by a dynamic polarity. All emotions are either positive or negative, either pleasure or pain, love or hate, security or fear, happiness or distress. If, therefore, we would find the psychological roots of this disturbance of the equilibrium, we must look for them in the emotional life of the individual.

The statement that the disturbance results from a negative emotion is not a complete or satisfactory explanation. We detest some circumstances in our lives and resent others which we know we cannot change without developing an equilibrium neurosis. When this neurosis develops, the contradictory emotions find a specific kind of outlet, frequently leading to this curious objection to the correct performance of a professional activity. These outlets can be designated as actions of the will. In order to understand this we must be able to separate the various mental dynamics and functions which play an important role in the same individual psyche.

Let us consider separately thought processes, emotions and violations and their inter-action in our individual life. In persons suffering from mental disease a distinction can be made among the various types of mental illnesses as regards the main source and character of the disturbance. A disease may be caused or centered in the functions of thinking. Cause and center of others will be found rooted in the emotional sphere; others again are centered in the sphere of will. We already have stated that equilibrium neuroses have their origin in the emotional sphere. These misdirected emotions cause queer will-actions and both together become the medium for pathology. Let us examine our cases from this angle:

The undertaker can no longer endure the attitude of constant mourning and tries to achieve an emotional balance by improper gayety and jokes. In order to compensate for his dissatisfaction he forces his jokes and his gaity to an unnatural degree and develops a strong link between joking and will power. He stresses gayety and joking until an uncontrolled reaction between his dissatisfaction and joking is created, which he is now unable to control, because a dynamic mental disorder has developed. It is the unnatural tie-up between emotions and violations which create this pathological state. The attorney becomes tired of enforcing the law. The emotional tie to his ideal as a good law abiding citizen is worn thin and, according to the violent disposition, he leans more and more towards an emotional demand for law breaking. In conjunction with volitional dynamics, this law-breaking tendency develops a severe affective demand for "sins and law-breaking". In the case of our attorney, the basis of the pathological conflict is a somewhat different situation. It is not the need for an emotional, conventional mourning gesture which leads to the breakdown, but the conflict between the volitional demand for law breaking and the rational intellectual dynamics which are his thought contents. The knowledge of overstepping legal boundaries and the volitional factors of doing so bring about the pathological conflict. In this equilibrium neurosis, we should be careful in differentiating the somatic equivalent because their dynamic backgrounds are different. This form of neurosis is complicated but is also instructive in the study and understanding of this type of psychopathology. The neurosis of the clergyman is similar to that demonstrated in the attorney. His conflict also is one between "sinful volitive actions" and intellectual settings.

The medical equilibrium neurosis requires more exhaustive discussion. Here again positive idealistic emotions are smothered by professional experience. The emotional conflict

results not infrequently in an ironic attitude rather than in a tendency to escape into drug addiction. Generally, irony cannot be considered a real pathological factor; we have not collected enough information about the role of humor and satire as a form of pathological reaction apart from Siegmund Freud's comments on jokes in his theoretical treatise. The form of joking which the undertaker adopted and which obtrudes itself in situations where mourning and seriousness would be required is a decisive pathological factor. A still more serious case is that described in Jarl Hammer's book. The possibility of positive emotional expressions of joy and happiness can be thwarted and even the dynamics of thinking can become disturbed by bringing them under the domination of a limited sphere of unpleasant, negativistic routine thoughts.

If we consider the professional equilibrium neurosis in the usual psychiatric aspect, we find that it may result in practically every form of pathology enumerated in present day psychiatric terminology. The most frequent mild form is a state of fear of various kinds; fear of being found out; fear of failure; fear of performing inadequately in one's profession; fear of losing a position, etc. Fear may also result in severe claustrophobia or agromania as well as any other form. Frequently these anxiety stages are called "breakdowns". Individuals with tendencies to melancholia and depression will progress in this direction, even to the point of suicide, as in the case of the clergyman. At times manias result when an attempt is made to develop a defensive attitude against the danger of falling prey to such a neurosis. Where the whole situation tends towards a dissolution of the inner structure of the mental personality, schizophrenia may ensue; paranoic trends will develop where the intellectual functions become diseased. Hemmer's doctor clearly shows such traits. The equilibrium neurosis has the same characteristics as any other mental disease to which we apply the social psychiatric viewpoint. The fundamental difference between social and individual psychopathology is that they represent two entirely different aspects of theoretical consideration,—the general pathological picture on the one hand and the different determining causes on the other. The individual's psychiatrically clearly defined disease may be caused by various social factors, or any potentially pathologic social situation may result in mental disease.

In the therapeutic aspect of professional equilibrium neuroses, considerations of individual and social psychiatric phases and their interrelation are of even greater importance. Mental illness caused primarily by social circumstances cannot be completely cured from an individual psychiatric angle. The casual conditions of any neurosis and its final cure are profoundly interwoven, as C. J. Jung has emphasized. If one becomes ill by reason of unfortunate professional conditions, particularly if the disease takes the form of an equilibrium neurosis, he will not be completely cured if the psychiatrist merely treats the "breakdown" or the depression neurosis as such, because no complete cure can be attained unless the casual relationship to his profession is investigated and remedied.

This, however, does not mean that individual psychiatry should not be applied if and when necessary, but the treatment must not lose sight of the wider social psychiatric view. Among the cases of professional equilibrium neuroses with which I have dealt, there was not one that developed into such a severe pathology that individual treatment had to be stressed. Where severe neurotic or even psychiatric conditions make up the somatic picture, there must be a period of convalescence which allows for the treatment from the social aspect. These are cases where the individual is not profoundly injured by professional misfortunes and every effort should be made to make him forget these circumstances, at least during the initial period of treatment. Even at this early stage the therapist should know how to solve the social psychiatric task. Close observation of the patient's reactions and an equally sincere study of his individual relations and attitudes towards his professional work should be undertaken from the start. This is essential to the carrying out of a successful course of treatment.

Even a mild form of equilibrium neurosis should receive combined individual and social therapy from the outset. Any pathological state of anxiety indicates a fear of something specific and any depression signifies that

the person is depressed by something, the "something" being the profession which has an important bearing upon in the whole process of disease and cure.

There are two alternatives in connection with the social therapeutic aspect: Whether the solution is in removing the individual from his professional sphere, or in the readaptation of the individual to his profession. Both procedures must be given due consideration. In mild cases, the second solution would not only be easier, but also better for the whole process of cure. In cases of equilibrium neuroses that I have observed, it is a sign of a profound disturbance if a readjustment is not possible. Real equilibrium neuroses do not occur frequently in the early stages of a professional career, but appear usually after years of activity in an occupational sphere. Therefore it is not a simple matter to develop a new professional skill and every endeavor should be made to induce a readjustment to the profession. Careful consideration should also be given to the question whether the negative emotions toward the profession arise from the profession itself or result from circumstances secondarily connected with it. Aversion toward the environment, colleagues, the "boss," or toward still more superficial factors may cause a negativistic attitude which eventually may turn into hatred of the occupation itself. In most cases of a severe equilibrium neurosis, this animosity has been extensively projected into the profession itself, so that the unbalanced individual would find it difficult to tell whether he hates the profession or the circumstances connected with it. As with the attorney, we sometimes find cases where the neurotic state has not resulted in a direct emotional negativistic attitude toward the specific profession itself. Nevertheless, his law-enforcing activities were the background of his pathological state and a cure could be attained only by clearing up the entire relationship.

The first step in a successful social psychiatric cure consists in making the patient conscious of the dynamic relationship which disturbed him so severely in his professional activity. Very often a major part of the fear and depression can be eliminated in this way. In mild cases, in which an adjustment toward the profession can be achieved, a proper outlet will have to be found for the tendencies causing a conflict between the individual and his profession. In order to balance the monotony of strictly routine professions, suggestions for the patient's leisure time may prove helpful. A growing interest in the progressive tendencies or the research side of this or that profession connected with the patient's sphere of interests may become a therapeutic factor.

Balancing the professional life of the patient by a more active stimulating social life is another way of helping to adjust the person. In this manner, I was able to solve the problem of the undertaker by suggesting to him that he direct his interest towards charity work, which provided a counter balance for his craving for misplaced gaiety and even provided a moral background for his activity as an undertaker. However, the balancing of emotional conflicts in themselves is not always the easiest way to follow. It is often of advantage to replace the negative emotion by another interest of a positive and higher quality. Such factors can consist in social interest, esthetic attitudes, etc. Such changes however, cannot be achieved or transferred to the patient merely by intellectual preaching. They can be made real to the patient only through concrete suggestions and repeated proof of their importance in his practical life. To achieve this is a task for the individual therapist.

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The Relief of Specific Psychiatric Symptoms by Convulsive Shock Therapy*

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I. *Introduction*

Following the early work of von Meduna in the treatment of schizophrenia with convulsive therapy came a period marked by many studies comparing the effect of metrazol convulsive therapy with insulin coma therapy. Most such studies were made on groups of patients selected on the basis of diagnosis. Within a short while, it became apparent that so-called "affective" psychoses appeared to respond better to metrazol than did the so-called "schizophrenias". Many criteria were eventually invoked in estimating results of treatment of these and other groups. These criteria of effectiveness of metrazol therapy could more properly be called criteria of effectiveness for any treatment. Electric shock convulsive therapy has shown the same effectiveness as metrazol on the symptoms studied in this report.

Age at onset, structure and stability of the pre-morbid personality, duration of symptoms, relations of stress situations and intensity of reaction all came in for important consideration in the determination of prognosis. The authors have accepted, as have most psychiatrists, the fact that the average psychiatric patient will have the best prognosis if the illness occurs in a fairly stable individual without outstanding evidence of previous disorder or predisposition, if it occurs in the third or fourth decade, if it has been of short duration, if there have been significant environmental stresses and if the symptomatic reaction shows a vigorous emotional response.

We recognized the artificiality of diagnostic categories and the fact that these categories, however well they may represent distinctive similarities of the patients, do not include sufficient of the above prognostic criteria to carry adequate indications for choice of treatment or for judging its prospective value. The present study was made on a diagnostically mixed group of patients who had been treated with metrazol at the Worcester State Hospital and on whom observations extending over a year or more following treatment had been possible. An analysis was made of the routine systematic descriptions of these patients recorded by over a dozen different psychiatrists. A group of twenty common, readily definable, easily distinguished symptoms were selected for study without regard to diagnosis. The response of patients to metrazol treatment was followed in terms of recovery from each of these particular symptoms. Intermediate degrees of response short of recovery were ignored for the purpose of this report.

A total of 133 metrazol-treated patients were studied. There were 72 males and 61 females. These consisted of 79 schizophrenics, 21 manic-depressives, fourteen involutional psychoses, six psychoneuroses, and thirteen patients with other psychiatric disorders. These patients had had from 4 to 49 convulsive treatments, the average case receiving 17. Each patient was given three convulsions weekly on alternate days. The termination of a course of treatment was predicated on a period of at least three treatments during which no noticeable change of any sort was seen in the patient. Within the ranges used the general results did not seem to be related to the number of treatments. Responses of patients in the few weeks just after the end of a course of treatment were noted as were responses recorded at least one year later. The average follow-up period was 17 months.

II. *Results*

The attached tables show the responses of the indicated symptoms to convulsive treatment in percentages of the initial number of patients presenting that symptom prior to this treatment. The status a year or more after completion of this treatment is sim-

*From the Worcester State Hospital, Worcester, Massachusetts.

ilarly given. Table I, Column 3, indicates symptomatic response soon after completion of metrazol therapy; Column 4, the condition at least a year following completion of the course of convulsive treatments. The trend towards continued improvement during the follow-up period averaged 16 per cent of all symptoms, the exceptions being suicidal attempts, untidiness and bizarre acts.

Table II deals with the schizophrenic patients among the mixed group studied. In this table, the same trends are observed but the trend toward continued improvement was less, averaging 10 per cent.

Table III shows the selected 20 symptoms in a control group of 111 schizophrenic patients admitted in 1936 before the "shock therapy" era. Their status one month after admission is compared with three and with 17 months later (after equivalent periods of hospitalization experienced in the metrazol treated group). The average rate of continued improvement after the third month in the control group is 9.5 per cent compared with 10 per cent in the treated group. The recovery rate for the selected 20 symptoms averaged

TABLE I
METRAZOL TREATMENT
MIXED DIAGNOSES

No. of Patients with Symptom before Treatment	SYMPTOMS	% Immediate Recovery after Completion of Treatment	% Retained Recovery after 1 to 2 years
19	Suicide Attempts	95%	79%
28	Refusal of Food	93%	96%
44	Untidiness	82%	77%
79	Bizarre Acts	75%	73%
37	Mutism	70%	81%
68	Depression	69%	81%
62	Anxiety	68%	79%
67	Overactivity	66%	79%
82	Hallucinations	62%	68%
55	Overtalkativeness	60%	73%
105	Delusions	57%	70%
74	Bizarre Speech	57%	61%
22	Confusion and Bewilderment	55%	95%
57	Underactivity	51%	65%
16	Disorientation	50%	88%
84	Lack of Interest	50%	56%
19	Overt Sex Acts	47%	68%
79	Inappropriate Affect	42%	54%
34	Euphoria	15%	62%
15	Poor Memory	7%	73%
133	TOTAL AVERAGE	58%	74%

TABLE II
METRAZOL TREATMENT
SCHIZOPHRENICS

No. of Patients with Symptom before Treatment	SYMPTOMS	% Immediate Recovery after Completion of Treatment	% Retained Recovery after 1 to 2 years
6	Suicide Attempts	100%	67%
12	Refusal of Food	92%	92%
27	Untidiness	85%	78%
52	Bizarre Acts	73%	71%
24	Mutism	67%	75%
31	Depression	81%	84%
26	Anxiety	85%	85%
38	Overactivity	71%	76%
55	Hallucinations	62%	65%
32	Overtalkativeness	53%	63%
67	Delusions	51%	69%
61	Bizarre Speech	53%	59%
15	Confusion and Bewilderment	73%	93%
37	Underactivity	49%	65%
10	Disorientation	60%	80%
56	Lack of Interest	50%	54%
15	Overt Sex Acts	47%	73%
68	Inappropriate Affect	43%	54%
23	Euphoria	17%	57%
9	Poor Memory	0%	67%
79	TOTAL AVERAGE	61%	71%

TABLE III
CONTROLS
SCHIZOPHRENICS NOT TREATED BY ANY FORM OF SHOCK THERAPY

No. of Patients with Symptom before Treatment	SYMPTOMS	% Immediate Recovery after Completion of Treatment	% Retained Recovery after 1 to 2 years
8	Suicide Attempts	50%	63%
23	Refusal of Food	52%	71%
23	Untidiness	44%	9%
70	Bizarre Acts	40%	44%
32	Mutism	53%	53%
53	Depression	57%	79%
42	Anxiety	43%	64%
50	Overactivity	38%	52%
87	Hallucinations	38%	52%
37	Overtalkativeness	32%	49%
102	Delusions	23%	38%
68	Bizarre Speech	38%	52%
29	Confusion and Bewilderment	52%	52%
51	Underactivity	47%	63%
12	Disorientation	58%	33%
91	Lack of Interest	10%	29%
18	Overt Sex Acts	61%	61%
94	Inappropriate Affect	17%	38%
19	Euphoria	16%	47%
17	Poor Memory	29%	41%
111	TOTAL AVERAGE	40%	49.5%

40 per cent in the first three months among the controls but the metrazol treated schizophrenics showed 61 per cent recovery during the equivalent (treatment) period. As was to be expected from initial considerations, symptoms of vigorous emotional protest, such as suicidal attempts, mutism, and refusal of food, responded best to convulsive therapy, whereas symptoms of weak resignation, such as impairment of thinking, lack of interest, and inappropriate affect, responded least. The immediate negative response of poor memory is, of course, the well recognized consequence of the treatment itself, a symptom which clears up in a few weeks or less.

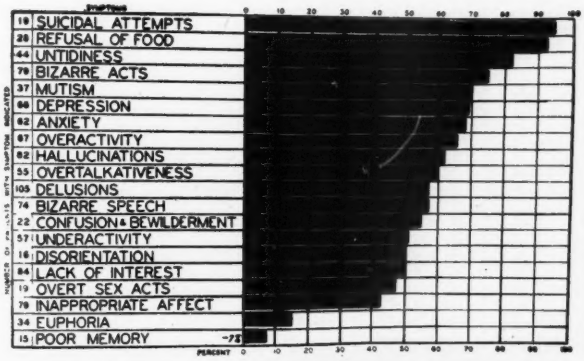
III. *Relation of Symptom Response to Diagnosis*

A further study was made of the relationship between diagnosis and recovery from the twenty specified symptoms. This study showed that general prognosis or diagnosis is not the determining factor for the immediate effectiveness of metrazol therapy on a specific symptom. The following figures are cited to substantiate this unexpected finding. Depression was immediately relieved by convulsive treatment in 64 per cent of manic-depressive patients generally considered to have a good prognosis, whereas 81 per cent of schizophrenic patients with a worse prognosis were promptly relieved of their depressed mood by treatment with metrazol. This suggested the generalization that the more predominant and constant a symptom, the less will convulsive therapy affect it. This is further supported by the finding that the persistent delusions of schizophrenics of the paranoid type were removed in only 32 per cent whereas 80 per cent of hebephrenic schizophrenic patients were relieved of their less dominant delusions. This is consistent with the diagnostic concept that delusions are secondary impermanent reactions in the hebephrenic but the primary reaction in the paranoid schizophrenic. Treatment produced no effect on the prominent lack of interest of the simple type of schizophrenic but yielded satisfactory results in 50 per cent of schizophrenic patients of other types. Our generalization breaks down when euphoria is considered. The constancy and dominance of euphoria in the manic-depressive of manic type was no apparent obstacle to recovery from this symptom, for the symptom yielded promptly in 43 per cent of manic-depressive patients as compared with only 17 per cent of schizophrenics with euphoria.

If one considers that euphoria is a primary reaction of the manic depressive patient, that is, a fundamental mood reaction, and that in the schizophrenic euphoria is generally secondary to the thought content, the above mentioned generalization would still hold. Refusal of food is favorably affected by metrazol therapy regardless of the diagnosis as is shown by 92 per cent recovery from this symptom among schizophrenic patients and 96 per cent recovery among patients with other diagnoses. Suicidal patients stopped efforts to kill themselves after their treatment course with the exception of one involutional patient, though involutional melancholia carries a favorable prognosis with convulsive therapy.

IV. *Prognostic Scale for Symptoms*

Charts I and II present a simple way to predict the expected immediate and late re-



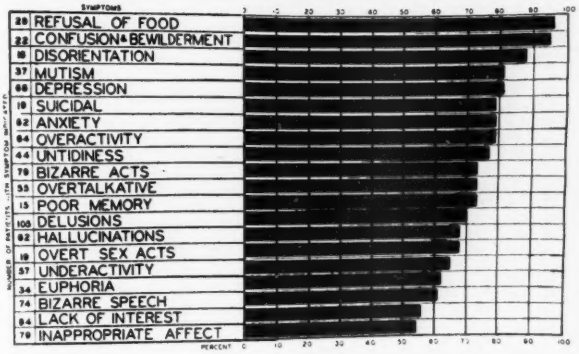


CHART I (above): METRAZOL TREATMENT
Percent Immediate Recovery From Symptom After Treatment.

CHART II (below): METRAZOL TREATMENT
Per Cent Late Recover One to Two Years After Treatment

sponse to convulsive treatment of 20 important symptoms regardless of the accuracy of diagnosis classification. In these charts the symptoms are arranged in descending order of the effectiveness of treatment. The presence of symptoms yielding most regularly to convulsive therapy serves as a criterion for recommending convulsive therapy. While these indications for undertaking convulsive therapy were established on metrazol-treated patients, they have since been extensively applied by one of the present authors (B. S.) during World War II to the treatment of several thousand military patients who were given either metrazol or electro-convulsive therapy. The results (to be published) showed no difference between response to electro-convulsive and metrazol convulsive therapy. In general this method of determining case selection for convulsive treatment served as the most satisfactory available means for estimation of the potential effectiveness of convulsive therapy.

Summary

It is recognized that a good diagnosis is dependent upon knowledge of the background and observation of the symptoms. Vigor of reactivity, background, type and duration of symptoms affect prognosis. Diagnostic disagreements are common; prognostic errors, very frequent. The effect of a specific therapy on the prognosis of a selected group is most difficult to determine, but the effect of a procedure on a specific symptom can be fairly readily established. By careful estimation of the effect of convulsive treatment on 20 important symptoms it is possible to more accurately foretell the degree of improvement to be expected than when diagnosis and general prognosis are the only guides.

Reflections of an Amateur Neuropathologist*

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The title of my address is "Reflections of an Amateur Neuropathologist" and I do not mean to be facetious for I am in truth an amateur, having pursued neuropathological studies for so scant a period as twelve years. Reflections should be made only after intensive study over a prolonged period but it is of some benefit to take inventory periodically and see in what direction one is heading. It is in this light that I reflect this evening as an amateur.

My introduction to the science of general pathology came from the late Dr. Richard H. Jaffé in whose laboratories there was more inspiration, spirit and life itself than can be found today on many hospital wards. To Dr. Jaffé, pathology was, as Webster defined it, the science treating of diseases, their essential nature, causes and development, and the structural and functional changes produced by them. Although he was a pathological anatomist par excellence, his knowledge of functional pathology was the basis of his greatness as a teacher and investigator.

To most medical students and to practicing physicians the word "pathology" conjures up visions of smelly autopsy rooms, bloody organs, pickled specimens in jars, microscopes and innumerable slides. To many, unfortunately, the term pathology is identical with morbid anatomical change and they recognize only two classes of disease: the anatomic, or organic, and the psychogenic. The symptoms of disease, however, are the result of altered physiology and it can be truly said that pathological physiology is the basis of medical science. Anatomical studies in themselves do not, in most instances, tell us the exact nature of disease. These studies must be paralleled with chemical, physiological, bacteriological, immunological and psychological investigations. The interrelationship of these many factors is so complex that one cannot be sure where one stops and another begins. The absence of morbid anatomical change does not indicate that a functional alteration has not occurred. In fact, Virchow emphasized that altered function invariably

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precedes anatomical change and that the former and not the latter was fundamentally significant.

The study of neuropathology is a tremendous undertaking and very few become truly masters of the subject. The neuropathologist does not exclusively study diseases of the nervous system, but rather diseases in which the nervous system is affected, for in the final analysis neuropsychiatry is today, as it was when it first became a specialty, a branch of internal medicine. The majority of the cases seen by the neurologist exhibit pathological physiology of the nervous system brought about by disorders which are basically non-neurological such as syphilis, tuberculosis, arteriosclerosis, arterial hypertension, vitamin deficiency, renal insufficiency, systemic intoxication, leukemia, Hodgkin's disease, etc. It is obvious, therefore, that a thorough knowledge of general pathology is indispensable to the understanding of diseases affecting the nervous system.

In general pathology it is not uncommon to see the pathological anatomist dissect the coronary arteries of a young adult who suddenly died from heart failure and attribute the death to one or two atheromatous plaques in a coronary vessel while in the next case, that of an aged individual who died from carcinoma of the rectum, the coronary vessels are extremely sclerotic and the myocardium is severely degenerated yet there were no signs of heart disease during life. Similarly, in neuropathology great discrepancies may be found between the morbid anatomical changes and the pathophysiological phenomena observed during life. The cerebellum may show extensive disease yet the patient may have no cerebellar symptoms. Although the thalamic syndrome is clearly recognized by all, large destructive lesions in the thalamus often fail to produce the classical symptomatology. Extensive softenings may be found in the basal ganglia yet the patients were free from the so-called extra-pyramidal disorders. Neuropathology has much insight to gain by remaining in close contact with general pathology.

There is an increasing tendency also to separate the inseparables: neurology and psychiatry. We have different local and national societies and different departments in many medical schools. This has been attributed in part to the great impetus given neurology by the surgical arm of the specialty. It is obvious, however, that the two are so closely intertwined that separation is virtually impossible. Many so-called psychiatrists have told me that psychiatry is no more closely related to neurology than it is to urology, dermatology or gastro-enterology. This is a gross misconception. We are all familiar with the havoc resulting in the mental processes from damage to the brain. The behavior disturbances and alterations in the thinking process in the post-encephalitic states, the mental deterioration in general paresis, Alzheimer's disease, Pick's disease and the post-traumatic cerebral states only serve to indicate that, although disturbances of behavior may accompany urological disorders, cystitis does not produce mental symptoms as uniformly or of the same order as encephalitis.

The interrelationship of psychiatry and neurology will, I believe, become even greater as our knowledge of the physiology of disease increases. Most neuropathologists are accused of being organicists when the causes of psychiatric disorders are considered. I must state at this point that I have the highest regard for the study of psychodynamics and psychoanalysis but I cannot always respect the opinions rendered by some of its practitioners. I even go further than many of the analysts in believing that psychogenic factors may be responsible for some forms of organic change in the nervous system through long-continued alteration of the physiological state.

The functional and anatomical integrity of the nervous system is greatly dependent upon its vascular supply, and emotional disturbances may precipitate migraine attacks, or even cerebral hemorrhage in susceptible individuals. Psychogenic factors, like viruses, bacterial toxins, and trauma, belong to the category of pathogenesis. It is unscientific to look at a histopathological state, be it hemorrhage, edema or malacia, and say, "This could not be psychogenic in origin." The neuropathologist must work in close harmony with the analysts just as he works with the immunologists, the specialists on allergy and the toxicologists.

Even the neurosurgeon of today is forced

to concern himself with psychiatric problems since he has been invited to operate on the brain in an effort to alleviate serious mental disturbances. A "block" or "choke" are no longer the prime indications for neurosurgery. Greater knowledge of the pathological physiology of the nervous system can come only from unified efforts of the clinical neurologist, the neurosurgeon, the neuropathologist, the neurophysiologist, the biochemist and the psychologist.

Many have said that neuropathology is all worked out and that neurophysiology is the road to be followed in the future. What they mean to imply is that morbid anatomic studies by means of the traditional technics will not be as fruitful in the future as they were in the past. This attitude is reflected in the neurological laboratories of today. Medical students, research fellows, residents, surgeons, and even psychoanalysts flock to the neurophysiological laboratories. Here the activity is great, the personnel numerous, the equipment complicated and expensive.

Only scattered students come to the neuropathological laboratories to pursue the morbid anatomy of the nervous system except near the date of a certification examination in Neurology, Psychiatry or Neurosurgery. Then they flock in by the score. Some try to create the impression that they are really interested in the anatomical preparations while others honestly admit that they want to study just enough to pass the board. Yet pathological anatomy is a necessary discipline in neurology as in general medicine. The organs must be examined after death and it is necessary that there be pathologists who can interpret their variegated appearances. Perhaps the easy things have already been discovered but there are still many problems unsolved. Moreover, the lesions produced in the experimental laboratory must be controlled and interpreted in the light of traditional neuropathology. The need for a knowledge of morbid anatomy of the nervous system will continue.

Comparative neurology will not be sufficient. The neurological disorders that afflict mankind rarely occur spontaneously in the cat, the monkey or the ape. Only the careful study of human neuropathological material can give the clinical neurologist insight into the pleomorphic manifestations of many of the diseases he discourses about so easily.

But morbid anatomy is not enough. Concentration of the attention on this aspect of the problems is apt to develop a rather hopeless attitude towards many of the neurologic disorders. In the heredo-familial degenerative diseases the destruction of the nervous system appears, and probably is, irreparable yet if the attention be shifted to the possibility that the degeneration of the neural parenchyma is the result of alteration in other organs of the body new possibilities of therapy, or at least of prevention, may be envisaged. The hereditary incidence of pernicious anemia has been described repeatedly yet treatment with liver extract serves to stop the course of the bone marrow disorder as it does the neural disorder. Wilson has emphasized that the degeneration in the brain in the disorder he did much to delineate follows, and does not precede, the liver disease. The general pathologist has been attacking the problem of hepatitis and liver cirrhosis and it may well be that the neurological disorder will be stopped in its tracks once the dynamics of the liver disease are elucidated.

The neuropathologist has a threefold responsibility: first, to the clinician by helping him understand the nature of the case he had followed so faithfully and many times so obscurely; second, to the student who is anxious to learn the natural history of many of the disease processes; and, third, to the science by adding to the common fund of knowledge. In pursuit of the first two aims, morbid anatomy will continue to be necessary, the science will not die, but in furtherance of the third aim, morbid anatomy is no longer sufficient. The neuropathologist of today is as proficient in experimentation as the physiologist and enters into immunological, pharmacological and biochemical investigations in the attempt to reproduce neurological disorders in animals which will point the way to their eradication in man. It is for this reason that today neuropathology is not limited to morbid anatomical studies. Only by the cooperation of the morbid anatomist, the physiologist, biochemist, immunologist and phychologist can light come to shine on many obscure disorders.

Neurological Clinical Pathological Conference

of the
Cincinnati General Hospital
CHARLES D. ARING, M.D., *Editor*

Case No. 78770

Presentation

A 34 year old, unmarried, colored man was admitted to this hospital on August 20 in a confused condition. A brother who lived with him said that he had been well until two weeks before when he was taken with drowsiness which steadily increased in severity until August 17 when he could not be fully aroused. The lethargy was followed by frontal headache, anorexia, nausea, vomiting, and confusion. He became careless of his personal hygiene. He had done extremely heavy labor up until the onset of illness; in fact the day before he had put 13 tons of coal into a basement.

There was a normal past history; three or four years previously while drinking he had been struck in the left eye by a thrown beer bottle and rendered unconscious for a few minutes. The eye was swollen shut for several days; there was no other complication of this injury.

At the time of admission to the hospital the patient presented striking mental abnormality. It was almost impossible to get in touch with him, he seemed nearly asleep; any movement appeared to be too much effort for him. For weeks he yawned frequently, on occasions he smiled. He would sometimes try slowly and slurringly to repeat a question; he seemed unable to answer any of them. His speech would become slower and slower and trail off.

Abnormal neurological findings were the following: the left pupil was 4 mm. in diameter, the right 2 mm., the latter appeared not to react to light, the left pupil reacted but not unless it had been stimulated by light for 20 to 30 seconds. He blinked at threat approaching from the left but not from the right. Abdominal reflexes were not obtained and the plantar responses were flexor on some examinations and extensor on others.

He continued drowsy and rather mute, totally detached from the situation. He would look when spoken to but rarely would say anything. He would try to cooperate in physical examination. He would be found with an extremity held fixedly up in the air in what should have been an uncomfortable position. The pupillary difference remained and the inconsistency of the plantar responses was noted in daily examinations.

On September 3 he maintained his extremities in catatonic postures for longer periods than previously. Smiles flitted over his face and sometimes he would laugh without apparent provocation. The abdominal reflexes were active and the plantar responses flexor in type. The pupils reacted slightly to light, the left was 4 mm., the right was 3 mm. in diameter. The diagnosis of schizophrenia was entertained.

An attempt at air encephalography (September 9) failed to fill out the spaces containing cerebrospinal fluid. In late September another attempt at air encephalography showed displacement of the ventricular system toward the right. He was operated upon in early October.

The mental reactions of the patient during the weeks prior to operation might best be termed as a sort of dreamy state. He would lie for hours unmoving, staring at the ceiling. His position would remain the same until an attendant would shift or turn him. His attempts at cooperating were slow motion personified and one would easily lose patience with him.

When he could be aroused he would accept food; he had to be fed, of course, and sometimes food had to be forced. Occasionally he would negativistically clamp his teeth against food. Sometimes he would become relatively alert but then he was confused and disoriented; he usually would "feel fine" during those periods, once he added "like fifteen cents". He was mostly incontinent of urine and stool. Rarely he would be found masturbating prolongedly.

Laboratory Data: The rectal temperature

was normal. A pulse rate of 70 was usual, though a rate of 55 to 60 was observed on twenty occasions, more frequently in the early weeks. The blood pressure was repeatedly registered around 105/65. Blood chemistry, counts, and blood Kahn tests were normal. Examinations of the urine were normal. Nothing abnormal was found in roentgen studies of the skull. On September 9 no air was seen within the cranial cavity after an attempted pneumoencephalogram. The air studies of September 24 demonstrated marked displacement of the lateral and third ventricles toward the right; the right lateral ventricle was considerably larger than the left. Despite the lumbar injection of 120 cc. of air there was none in the cisterns or in the subarachnoid spaces.

Cerebrospinal fluid studies were as follows:

Date	*Pressure mm. H2O*	*Color*	*Cells*	*Protein mg. Pct.*
8/20	300	Water	0	. .
8/26	200	"	0	43
9/9	Encephalogram	"	0	30
9/24	Encephalogram	"	0	31

Colloidal gold curves and Wassermann reactions were normal in each of these fluids.

Differential Diagnosis

Dr. I. Mark Scheinker: The clinical history is composed of two principal groups of signs: mental and neurological. The initial mental symptoms are listed as steadily progressing drowsiness and confusion. I stress the word initial because it seems that the patient was well until two weeks before admission. In neurological diagnostic consideration, the first symptoms are at times the most important. What occurs later may be the result of complications such as edema and hydrocephalus. The fact that the clinical diagnosis of schizophrenia was eventually entertained may indicate that the mental symptoms in this case were more marked than in the usual case with increased intracranial pressure.

The neurological symptoms were relatively few and were consistent with those sometimes accompanying increased intracranial pressure. Signs which might be interpreted as focal in nature were unequal and poorly reacting pupils, and the vague indication of right hemianopia.

The laboratory findings established the intracranial hypertension. The air encephalograms indicated marked displacement of the lateral and third ventricles toward the right.

The diagnosis of the underlying pathologic process seems relatively simple; we are dealing with a cerebral tumor, the localization of which is difficult. I believe that subdural hematoma may be ruled out in view of the absence of the story and evidence of recent trauma. What trauma is recorded occurred three to four years before he became ill. It seems safe to eliminate trauma as an etiological factor because of this long interval.

If this is a brain tumor, let us try to determine the localization. As has already been mentioned the leading symptoms were those of mental character. What is the value of mental disturbance in localization, if any? There are different schools of thought on this subject. There are those who believe that mental symptoms have localizing value and those who deny it. I am one of those who believe that mental disturbances of a certain character have localizing value, provided they occur without hydrocephalus and edema, and without high pressure, and if they are initial and leading symptoms. In any other case I would question their localizing significance.

Mental disturbances that are somewhat characteristic of frontal lobe tumor are lack of spontaneity and drive, emotional indifference, slow mental reaction, infantile or motiveless behavior, the so-called "joking ailment" ("Witzelsucht"), and a sense of well-being (euphoria) due to failure of insight, and apathy and indifference particularly to things affecting the patient himself. Often those patients who were formerly neat and careful about their appearance become untidy and careless, which may progress to the point where the patient urinates and defecates in bed or in public.

While any and all of these mental symptoms are frequently observed in frontal lobe tumors, especially in cases with bilateral lesions, none is pathognomic or specific. Stress may properly be laid on personality change and disorder of conduct provided they appear early and in the absence of signs of hydrocephalus.

A few words concerning sleep. Lethargy can be caused not only by encephalitis but also

by tumors, particularly those in the posterior end of the third ventricle or in the periaqueductal region. In rare cases sleep might be a symptom of value for localization. Lethargy and drowsiness are also seen in frontal lobe tumors. The patient whose lethargy is caused by a lesion about the third ventricle may be roused to clarity, whereas frontal stupors are changed to turbid awareness by stimulation. This differentiation might be of certain value in our case, when the patient was aroused he wasn't particularly bright.

The neurological symptoms of frontal lobe tumor you know and I can pass over them quickly, motor dysphasia, disturbance in the reflexes, and disturbed olfaction and vision if the tumor is inferiorly located. None of these focal signs were present in this case.

Glioblastoma multiforme and meningioma make up about 95 per cent of the tumors in this locale. Glioblastoma multiforme usually originates from the centrum ovale or corpus callosum, and meningioma from the base or the falx.

I am not inclined to discard the mental symptoms completely. I think they are important in this case. On the other hand, we have symptoms which indicate involvement of certain of the basal ganglia. He maintained cataleptic positions and he had spells of what may have been forced laughing. Also I think of the basal ganglia because tumors there in a position close to the lateral ventricle might show few clinical manifestations unless the foramen of Monro was occluded causing hydrocephalus. We have to consider a tumor which might have remained latent and thence rather quickly led to hydrocephalus. I believe that the tumor is localized between the anterior portion of the basal ganglia and the frontal lobe, close to the ventricle. I feel that a temporal lobe tumor on the left side would produce many more symptoms, particularly dysphasia or discharge phenomena; lesions in the anterior and mesial portion of the temporal lobe usually do not produce hemianopia though those of the posterior aspect of the temporal lobe may do so.

The tumor is probably in the posterior aspect of the left frontal lobe compressing the lateral ventricle; it might be a glioblastoma multiforme that originated from the corpus callosum.

Dr. Charles D. Aring: I would be inclined to agree with what Doctor Scheinker said about mental signs. Almost any mental sign that has been described in frontal lobe tumor may be seen with pneumonia, typhoid fever, or disease of the liver, and in fact with any prostrating disease. It is difficult to localize cerebrally on mental symptoms alone, since they are so dependent on the organization of the personality prior to the physical disease. If they are initial signs they must be weighted a bit more; I also agree that if both frontal lobes are involved one may see a facetious, euphoric, and uninhibited individual.

Student: Did the patient have hemianopia?

Dr. Aring: I am inclined to believe that he did.

Discussion of Pathology

Dr. Joseph P. Evans: With the impression that we were dealing with a tumor despite the improvement in the patient's condition, he was prepared for operation and on October 11 a left occipito-parietal osteoplastic flap was reflected. When the dura was exposed it was found to be extremely vascular. With the plan of taking a cerebral biopsy, an opening was made in the dura over the parietal lobe. It then became evident that we were dealing with a subdural hematoma. The dura was reflected and separated from the sac. The content of the sac was partly solid, partly fluid; obviously the hematoma was old. The sac was adherent to the dura but a line of demarcation was found readily. The entire subdural collection was removed.

Microscopic examination revealed a moderately thickened dura to which the incomplete envelope was firmly attached. The outer layer of the sac was made up of a thick layer of fibrous tissue (heavily stained for collagen), in which there were found many vessels which tended to run at right angles to the fibers. Most of these vessels were small, but some of the large sinusoids described by Leary[1] were present. Scattered throughout were collections of pigment-laden phagocytes, some of them in large clumps. The membranous envelope surrounded a cavity from which the blood had been evacuated. Toward the periphery of the hematoma the inner membrane was thick and was

not unlike the outer membrane; it thinned out as it approached the more central portion of the specimen. On the under surface of the membrane there was a thin layer of endothelial cells. The central portion was devoid of capillaries, sinusoids, and phagocytes, and was made up only of parallel bands of collagenous fibers.

The lining of the sac itself was an incomplete endothelial layer. Where the lining was incomplete the sac tissues were in immediate contact with disintegrating red blood cells and the connective tissue reaction appeared to be identical with that surrounding blood injected into cerebral tissue. This represented, we believe, the reaction of tissue to recently extravasated blood and was an indication of continued bleeding.

It seems likely that blood between the outer and inner membrane breaks down increasing the osmotic pressure within the sac, drawing fluid therein,[2] possibly across the arachnoid.

We want to emphasize the sequential developments of this case, so I shall read you the note written on November 4, shortly before he was discharged, which gave the first indication of any trauma. "He now gives a history of a blow in the left eye with a bottle, three or four years ago, while drinking. He says he was unconscious for a few minutes, then developed a badly swollen eye which remained closed for several days. He did not suffer headache thereafter or photophobia. There was no history of hemianopia, this he first appreciated about one week ago after it was demonstrated on many occasions." Doctor Aring incorporated this history of trauma in the protocol, but we did not obtain it until three weeks after operation.

On February 15, when he attended the Out-Patient Clinic, the following note was made. "He now gives the story that two weeks before coming to Cincinnati on July 7, he was struck over the right eyebrow by a piece of rock while mining coal in West Virginia. The wound bled a little. He did not lose consciousness and paid no special attention to the injury. This story is additional to that of injury some years earlier, when he was struck by a beer bottle. The histologic structure of the hematoma suggested that the hematoma was older than three months, but this may well be open to debate." Visual fields done on February 7 showed homonymous hemianopia with sparing of macular vision.

In subdural hemorrhage the hemianopia is usually on the basis of hemorrhagic infarction of the region supplied by the posterior cerebral artery of the same side. The mechanism is believed to be somewhat as follows:

The swelling hematoma sac causes a displacement of the hemisphere to the opposite side. Quite possibly local interference with circulation immediately subjacent to the expanding hematoma leads to edematous changes in the apposing cerebral tissue. In any event the uncus of the temporal lobe becomes displaced and the brain stem is shifted. There results from the brain stem shift a stretching of the inelastic third nerve on the side of the clot. This tautens the nerve and causes a reduction in flow through the posterior cerebral artery, at the point where it curves around the third nerve. Reduction of flow in the distribution of the posterior cerebral artery is of itself not enough to cause hemorrhagic infarction of the lobe as has been shown experimentally. It does, however, cause swelling of the lobe and the compounding of swelling hematoma, local circulatory changes beneath the hematoma, and reduction of flow in the posterior cerebral artery results in sufficient interference with the venous outflow from the lobe, that hemorrhagic infarction results. This is essentially perivenous in character, a characteristic of infarction which was first indicated to us by Doctor Scheinker.[3]

The most important lesson to be learned from this case is that subdural hematoma like systemic syphilis is a great imitator. You never can tell when you are going to meet it. It mimics many neurological disorders, as syphilis mimics many other diseases. The only way to make the diagnosis of subdural hematoma is to be continually aware of the possibility and, when the symptoms and signs are suggestive, to explore for it.

Clinical Diagnosis

Infiltrating tumor, left occipital region.

Doctor Scheinker's Diagnosis

Glioblastoma multiforme, originating from the corpus callosum and infiltrating left fronto-parietal region.

Post-Operative Diagnosis

Subdural hematoma, left fronto-temporal parietal region.

Dr. Kenneth Colby: Did the pupillary disturbance remit after operation?

Dr. Evans: The pupillary inequality persisted.

Dr. Scheinker: Would you consider the possibility of subdural hematoma with a history of trauma three or four years prior to clinical manifestations?

Dr. Evans: Frankly I believe the answer is no; though the literature contains cases in which there were time intervals of many years between trauma and diagnosis.

Dr. Aring: The patient couldn't give his story and the brother didn't know much about it. If we had related the entire history in the protocol, the case would have been nowhere as useful in pointing a most effective lesson. It is instructive to note that it took four months for his retrograde amnesia to clear.

As Doctor Evans has said, subdural hematoma is the great neurological imitator which may occur at any age from infancy onward. The last case of subdural hemorrhage presented here was 75 years of age and no one had suspected the correct diagnosis.

Student: Wouldn't you expect external ophthalmoplegia as a sign of third nerve involvement as well as pupillary dilatation?

Dr. Evans: In the monkey it has been shown[4] that the introduction of saline into the subdural space through a tap fitted into the skull results in a sequence of third nerve dysfunction of which the appearance of ptosis is first. Dilatation of the pupil next occurs and extraocular palsies develop last. In the patient, ptosis, if not specifically sought, is apt to be overlooked, and the patient's depression of conscious level may be such that extraocular palsies can not be readily demonstrated.

Dr. Aring: It should be said that one may see any one sign of dysfunction of the third nerve without the others being present. The less experienced we are the more we want the syndrome to be complete.

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Electric Shock Therapy

(The Design and Use of a Simplified Apparatus*)

JAMES S. L. JACOBS, M.D., and
WARREN E. GILSON, M.D.
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The present report describes the design and use of an electric shock apparatus which was constructed according to criteria set up after considerable experience with this form of therapy. Standards were defined in terms of safety, simplicity of use and construction, the ability of the device to deliver a constant current and variable durations of its flow, small size and low weight. It was also considered desirable that the apparatus be completely self-contained, holding necessary electrodes, head band, etc. The apparatus (Fig 1) was designed and constructed by one of us (W.E.G.) and employed, in clinical practice by the other (J.S.L.J.).

In terms of safety, the machine was so designed that in case of failure of almost any component, the current is automatically turned off. In addition, release of the "treatment" button, at any time, breaks the current. It was determined, in preliminary series, that

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a more than sufficient number of variable treatment doses could be obtained with one constant current, using a calibrated series of shocks by means of an electronic timer. This obviates the need of two variables and requires the setting of only one dial (timer) to the desired duration —0.4, 0.6, 0.8, 1.0, 1.2, and 1.5 seconds. This control, the current flow meter and the main on-off switch, with its adjacent power pilot light, are attached to the front panel and are, therefore, in constant view of the operator. The front panel contains another protected lamp which lights only during the application of the shock current. Release of the "treatment" button at any time, cuts off the current as well as this lamp. The device contains a reliable current regulator maintaining an output of 400 m.a. throughout any treatment notwithstanding the well-known wide variations of skin, scalp and skull resistances. This factor has the added advantage of removing the necessity of using the commonly employed variables of current regulation. The size of the machine is 7¼ x 8¾ x 8½ inches and weighs 15 pounds. A storage compartment with a sliding door contains the electrodes, head-band, flax, etc.

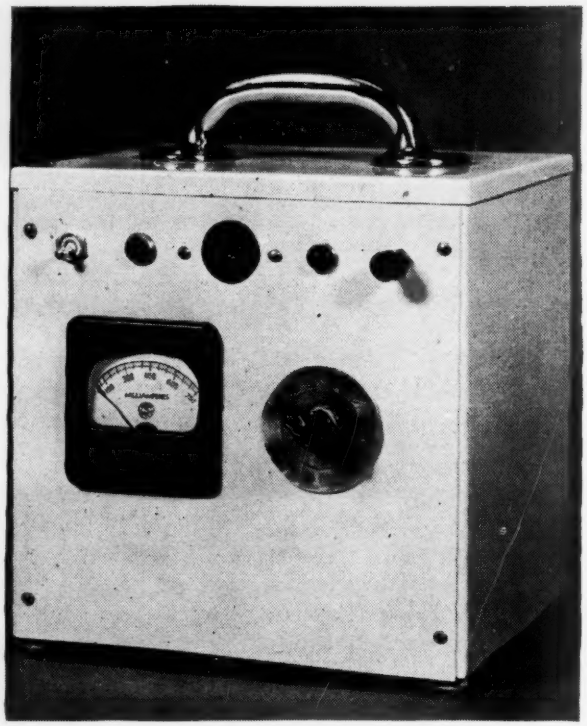

FIGURE I

Photograph of the electric shock apparatus. Controls, upper row. Left to right: On-off switch; power pilot light (green); output to patient (socket); treatment pilot light (red); treatment switch (push-button).

Lower row. Left to right: Current meter; time switch (duration of current dose), in seconds.

The lower three inches forms a space for electrodes, flex, etc., and is accessible through a sliding door in the back of the apparatus.

The therapeutic results obtained with this apparatus, in 500 treatments, have not differed from those obtained with two other electroshock devices with a similar group of patients. However, several independent observers have corroborated our impressions that not only were there fewer cases of respiratory embarrassment noted following treatment with our machine, but also the initial tonic contraction after application of the current was somewhat milder. It is possible that both of these improvements were due to the use of a constant current throughout the treatment, without the usually obtained initial peak and gradual reduction as polarization occurs.

Our method of administering electroshock therapy may be described as follows: Breakfast having been withheld, the patient receives sedative premedication consisting of small doses of a rapidly acting barbiturate (one to three grains of sodium amytal, or one and one-half to three grains of seconal) with one grain of codeine, by mouth. Agitated or disturbed patients are given commensurately larger doses of sedative; as much as 8 grains amytal will at times be given intravenously prior to treatment. Not only have no ill-effects followed such premedication, but it has been noted that the patients have fewer undesirable behavior reactions or symptoms following treatment. The convulsion is produced after curare has been administered intravenously. The patient lies supine on a bed, a blanket roll having been placed across the back at the level of the inferior angles of the scapulae. He is then covered with another blanket, although any other insulating material may be employed. Four assistants hold the patient as follows: two apply pressure over the anterior superior spines of the iliac bones and over the knees; one grasps the hands and applies pressure over the shoulder joint of that side; the remaining assistant places one hand under the pillow supporting the patient's head, so that it rests under the occiput, the other hand holds a padded tongue blade, which has been inserted

between the patient's molars; this hand also supports the jaw in order to prevent over-extension of the temporomandibular joint and possible dislocation. In this manner, the patient is held in optimal extension throughout the convulsive seizure and complete control is maintained over the head and cervical regions.

It has been found that respiratory embarrassment, following an electric shock treatment, depends upon several factors. The most prominent is physical constitution. Certain individuals, especially those of sthenic and plethoric habitus, when in middle age, are prone to develop post-ictal respiratory difficulties. These are unrelated to curare dosage and are due to both pharyngeal and laryngeal obstruction. A similar reaction pattern in patients of this constitution is well-known to anaesthetists particularly is it evident with electric convulsive treatment, if the patient is tense immediately prior to therapy, a further indication for sedatives. Delayed resumption of respirations occurs occasionally in debilitated involutional psychotics, especially males. This is often due to over-dosage with curare, to which they sometimes exhibit increased sensitivity. The former type of respiratory difficulty is quickly controlled with the application of an oxygen mask and artificial respiration. The latter complication responds to the administration of prostigmine. The occurrence of either type of delayed respiration is uncommon in our experience and it is seldom that such manipulations are necessary.

A third type of complication occurs when the operator applies insufficient current to produce a grand mal seizure. In this case, the cumulative effect of the respiratory depressant electric current may, after a grand mal seizure has finally been obtained, produce a prolonged apnoea. This complication responds satisfactorily to oxygen and artificial respiration. It is our conviction that there are several disadvantages and no advantages in the use of too small convulsive dose. The petit mal seizure has no therapeutic effect, it is followed by an increase in amnesia and confusion and, as noted above, if followed by a therapeutically effective grand mal seizure, occasionally produces respiratory difficulties in addition to these undesirable sequellae. As soon as a petit mal seizure is suspected (it will usually be indicated by an inspiration, swallowing movements or motor restlessness), a second shock, from one to one and one-half seconds in duration, should be applied immediately. It is our impression that, in general, respirations are resumed in a direct ratio with the speed at which a grand mal seizure is obtained. This latter factor is usually related directly with the duration of the current dose.

In general, schizophrenics and younger patients having other psychoses, require a shorter current dose than do older patients, especially those of the involutional group. In order to avoid the production of petit mal seizures, it is advisable to increase the dosage by about two-tenths of a second after each three or four treatments. However, many patients require a more rapid advance than this. Also, the immediate premedication must be considered in the choice of time dosage. Usually, an initial dose time of 0.8 seconds will provide a wide margin of safety in order to avoid petit mal seizures. The frequency of administration and the required number of electric shock treatments is a highly individualized matter, which has been discussed in detail elsewhere and is beyond the scope of this presentation.

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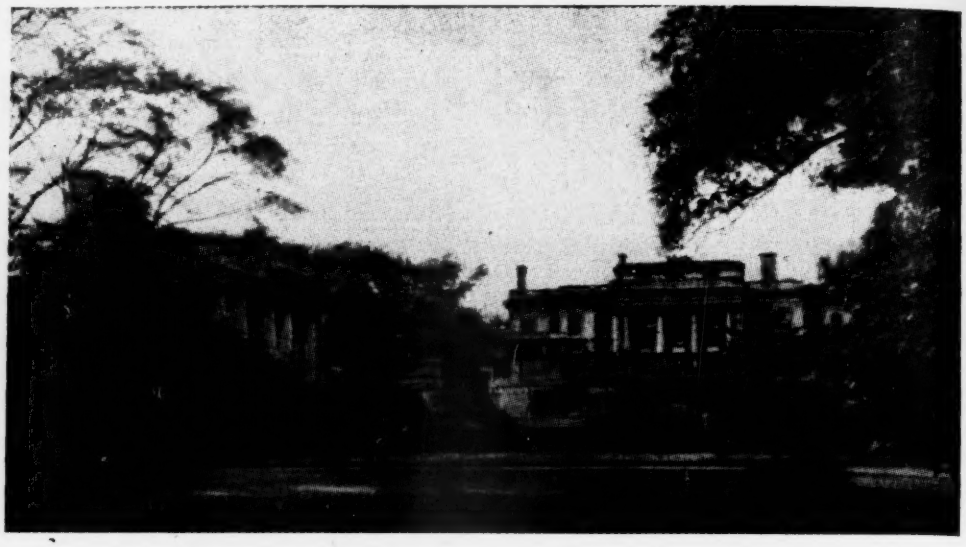

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